60 cells through activation of caspase in conjunction with cytochrome c release induced by a processed product of Bid. Now we are further investigating the relationship with the mitochondrial potential, ROS and Fas expression.

[PC1-13] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Intermedeol-Induced Apoptosis Involved Fas/Fas-L and cytochrome c dependent pathway in Human Leukemic cell HL-60

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We previously demonstrated that Intermedeol, a sesqueterpene isolated from Ligularia Fischery var., had a antitumor activity by induction of cell differentiation and apoptosis in HL-60. In this study, we examined signaling pathways implicated in Intermedeol up-regulation of Fas receptor expression and caspase 8 activation of Intermedeol. Bid is processed after Fas ligation and thus might activate the mitochondrial-dependent apoptotic cascade. Activated Bid preceded the release of cytochrome c without mitochondrial permeability transition. Cytochrome c release led to the activation of caspase 9 and downstream death effector, caspase 3. These finding suggest that Intermedeol induced cytochrome c-dependent apoptosis through Fas/Fas-L pathway.

[PC1-14] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Requirement for JNK activation in costunolide-induced apoptosis

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Costunolide is an active compound isolated from the root of Saussurea lappa Clarks, a Chinese medicinal herb, and considered as a therapeutic candidate for various types of cancers. In this study, we investigate the effects of costunolide on the induction of apoptosis in human leukemia cells and its putative pathways of action. Using diphenylamine and Hoechest apoptosis analysis, costunolide caused apoptosis of U-937 cells in a concentration- and time-dependent manner. Since costunolide-induced apoptosis was completely prevented in Bcl-2 overexpressed cells, these apoptosis was associated with Bcl-2, Furthermore, we demonstrated a requirement for c-Jun N-terminal Kinase, a member of the mitogen-activated protein kinase family in mediating cosunolide-induced apoptosis of human leukemia U-937 cells. JNK activation by costunolide contributed to apoptosis because transdominant-negative JNK significantly blocked costunolide-induced cell death. These findings cause the possibility that the JNK activation by costunolide can inhibit the Bcl-2 activity by phosphorylation.

[PC1-15] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Studies on the Growth-Inhibitory Effects of Pini Resina and Sodium Chloride against Oral Bacteria

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There has been considerable interest in the use of antimicrobial agents including a number of antiseptics, antibiotics and some natural products, as additives of some oral hygienic products for the purpose of treatment and/or prevention of periodontal disease.